Eur J Vasc Endovasc Surg **22**, 381–382 (2001)
doi:10.1053/ejvs.2001.1460, available online at http://www.idealibrary.com on IDEAL®

CASE REPORT

Late Axillo-brachial Arterial Aneurysm Following Ligated Brescia–Cimino Haemodialysis Fistula

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Introduction

John Hunter first described the development of arterial dilatation proximal to longstanding arteriovenous fistulae in 1757.[1] Late proximal arterial aneurysm secondary to ligated traumatic arterio-venous fistulae are a rare, but documented complication.[2–6] We present a unique case of pan-arterial aneurysm formation discovered 8 years after closure of a Brescia–Cimino fistula, managed with a bypass graft. There have been no previously recorded cases described in the English literature.

Case Report

A 51-year-old gentleman presented in February 2000 with a 3 week history of increasing swelling in his left axilla. He also complained of an associated dull ache in the left shoulder and tingling in all his fingers on that side. In 1974 the patient had developed membranoproliferative glomerulonephritits. He subsequently had a left Brescia–Cimino fistula formed for haemodialysis in 1976. A year later he had his first renal transplant. A second transplant followed this in 1981 for graft failure. His second renal transplant continues to function well. In 1992 the patient underwent routine surgical excision of his patent left Brescia–Cimino fistula for cosmetic reasons. Angiography at that time was normal.

On examination he was normotensive but had a prominent ulnar, brachial and axillary pulse with a palpable supraclavicular thrill. There were emboli in the ulnar digital nail beds. Allen's test revealed an absent radial in flow to the hand and there was no Doppler signal over the radial artery at the wrist. There were no neurological abnormalities. Selective digital subtraction upper limb arteriography (Fig. 1) showed markedly ectatic and tortuous subclavian, axillary and brachial vessels. The ulnar artery was patent, with an occluded radial artery at the site of the previously ligated fistula. The paraesthesia was thought to be caused by pressure on the brachial plexus.

Surgery was indicated on the basis of the size of the aneurysm, neurological symptoms and concern over embolism of his only run-off vessel. At operation the subclavian artery was exposed through an infraclavicular incision and the distal brachial artery was exposed in the antecubital fossa. Harvested left long saphenous vein was anastomosed proximally end to side to the subclavian artery which was ligated distally. The vein graft was tunnelled in a subcutaneous route to the recipient site. The origins of both terminal vessels were involved and they were disconnected separately. A section of aneurysmal artery was sent for histology before proximal over-sewing. An end to end anastomosis was carried out to the dominant ulnar artery. The radial artery was then anastomosed to the vein graft and there was good flow through the graft following this.

Postoperatively the patient made a full recovery. Subsequent duplex scans have demonstrated a patent graft with no evidence of stenosis. The aneurysm has thrombosed and there is no filling from the circumflex humeral artery. Histology showed disruption of the normal vascular structure with complete fragmentation of the elastic laminae consistent with aneurysm formation.

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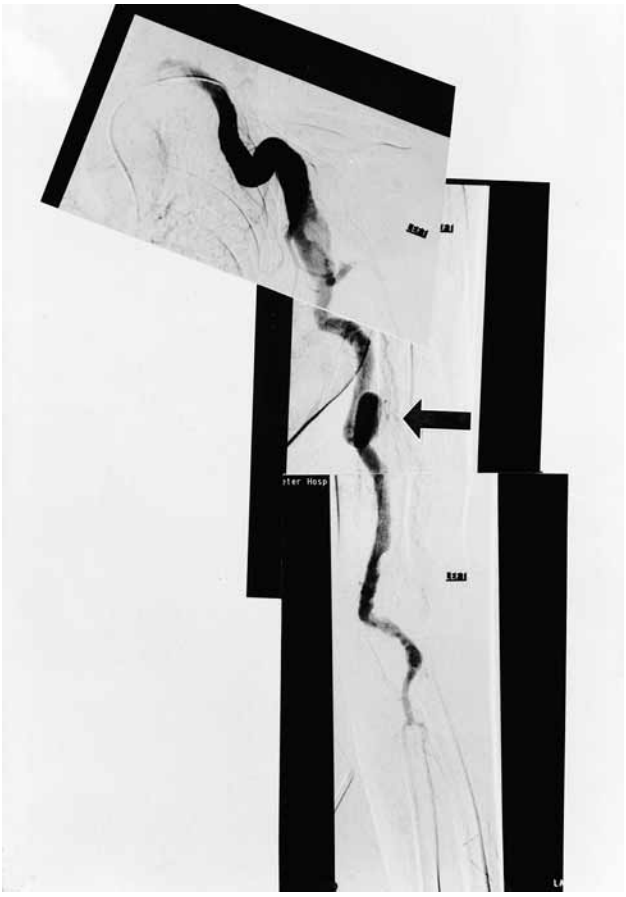

Fig. 1. IA-DSA demonstrating pan-arterial aneurysm with 5 cm diameter (on duplex) mid-axillary component. The radial artery is occluded. Note the brachial artery loop (arrow).

Discussion

Progressive arterial dilatation proximal to a ligated fistula is rare. All such cases in the literature refer to longstanding traumatic arteriovenous fistulae that were closed and observed to undergo subsequent aneurysm formation 1–15 years later.[2–5] The most common vessels involved were the superficial femoral artery and iliac vessels. We found only one case of upper limb aneurysm formation secondary to a traumatic fistula.[6] Our case is the first documented report of late proximal arterial aneurysm after closure of a haemodialysis fistula.

The mechanism of this "vascular remodelling" is postulated to be related to the increased flow in the vessel, which results in increased shear forces. Longstanding high flow induces transverse tears in the elastic fibers of the internal elastic membrane, progressing proximally from the site of fistula.[7,8] Dilatation may be mediated by a humoral agent, secreted and acting locally. Endothelium derived relaxing factor, secreted by the vascular endothelium and stimulated by the increase in frictional force has been implicated.[10] This may explain why the entire arterial segment under increased flow dilates rather than the collateral vessels.

The onset of secondary dilatation after closure of arteriovenous fistula is unpredictable. If left untreated, two main complications can occur; embolism or rupture. This report highlights the potential need for duplex surveillance of patients who have undergone closure of longstanding traumatic or iatrogenic arteriovenous fistula, particularly if new symptoms arise. Those who develop aneurysms should be offered exclusion bypass grafting and should be monitored.

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Accepted 5 July 2001